Beyond Skeptical Relativism: Evaluating the Social Constructions of Expert Risk Assessments

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Constructivist analyses of risk regulation are typically agnostic about what should count as robust or reliable knowledge. Indeed, constructivists usually portray competing accounts of risk as if they were always equally contingent or engaged with different and incommensurable issues and problem definitions. This article argues that assumptions about the equal reliability of competing accounts of risk deserve to be, and sometimes can be, examined empirically. A constructivist approach grounded in epistemological realism is outlined and applied empirically to a particular comparative U.S./U.K. case study of pesticide regulation. The article argues that while the scope for interpretative flexibility when addressing risk issues is clearly extensive, it is not unconstrained. By scrutinizing the structure and coherence of particular risk assessments and policy decisions by reference to both empirical evidence and commonly held robust standards of interpretation, the article argues that the U.K. evaluation was not only less precautionary than its U.S. equivalent, but it was also less well constructed and therefore less reliable. Several social and institutional characteristics of U.S. and U.K. policy making are highlighted that appear variously to facilitate or inhibit the production of reliable knowledge and the making of prudent policy decisions.

In recent years, social constructivist analysts have endeavored to provide a rich and hopefully policy-relevant understanding of the role that scientific experts can and do play in policy-making processes and to explain why experts often disagree about risk (Jasanoff 1987a; Schwarz and Thompson 1990; Royal Society 1992). Constructivists have argued that a range of prior social, cultural, institutional, and practical commitments are embedded and reproduced, usually tacitly, within technical risk assessments that typically purport to be objective. Such commitments shape vital intellectual decisions, for instance, about the legitimate and relevant dimensions of a risk problem; the preferred paradigms of analysis; the criteria for certifying and interpreting evidence; and the ways in which different types of uncertainty are

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recognized, engaged with, and represented. Building on those insights, a substantial body of empirical work has explored the wider social and environmental implications of dominant institutional constructions of risk, much of it providing rich insights into this crucial arena of government policy (Jasanoff and Wynne 1998).

There is, however, a tension in much of this literature. Most analysts proceed programmatically to support Bloor's (1976) injunction to take an approach that is symmetrical and impartial with respect to the truth value of the beliefs being deconstructed. Methodologically, scientific disputes are analyzed as if interpretative flexibility were unlimited, or at least constrained only by social and cultural conventions (Radder 1992). From such a perspective, concepts of validity, for example, are effectively represented as being entirely matters of social judgment. Nevertheless, some constructivists acknowledge that the social character of knowledge is such that it can sometimes successfully identify and comprehend real and extra-discursive objects and phenomena. In other words, they sometimes indicate, at least in principle, that the material agency of nature is, or may be, a constraining factor on what scientists (and others) believe (Hannigan 1996; Jasanoff and Wynne 1998). Constructivists with realist inclinations sometimes refer to the natural world and to material reality when providing a preliminary or provisional gloss to what are essentially sociological accounts, whereas others acknowledge their realism by using terms such as *weak constructivism* or *moderate constructivism* (Chubin and Restivo 1983). Most sociological analyses of risk assessment are, however, at least implicitly, if not explicitly, agnostic when it comes to questions about what should or does count as reliable knowledge.

The important question that has not yet been addressed is whether the agnosticism that may be methodologically appropriate in advance of a social deconstruction of a scientific risk assessment always remains appropriate once that deconstruction has been accomplished. In this article, we argue that one of the lessons to be learned from the practical utilization of the sociology of scientific knowledge is that some risk assessments are far more robust and better constructed than others. Policy judgments based on shoddily constructed risk assessments are more likely to run into trouble than those based on more robust ones. The history of the bovine spongiform encephalopathy saga illustrates that thesis, but in this article we will describe an interesting and important area of risk assessment that is rather less well known. Using a cross-national case study on pesticide regulation, we argue that an assumption of the equal (un)reliability of expert risk analysis deserves to be, and can be, examined empirically and illustrate why those empirical studies can ex post justify the abandonment of agnosticism concerning the existence and

nature of some risks. That is, however, a bold claim because it is tantamount to claiming that sociological study of science can enable social scientists to make a positive and substantive contribution to scientific discourse, highlighting matters that members of the scientific and policy-making communities may themselves be unwilling or unable to acknowledge.

An Analytical Approach

Our approach involves rejecting the methodological (and epistemological) relativism of most constructivist analyses and instead presupposing a conceptual approach based on epistemological realism (Bhaskar 1978). Many analysts give the impression that the only alternative to skeptical relativism is an outdated scientistic view of science (Richards and Martin 1994), but realism is quite distinct from the scientistic epistemologies underlying conventional approaches to studying risk controversies. In particular, realists do not claim that science possesses a universal and ahistorical methodology that guarantees true knowledge, although they can acknowledge that historically contingent methods and strategies for constructing reliable knowledge have been developed in practice (Chalmers 1990). Realist analysts can acknowledge that social factors affect all facets of scientific activity but are not compelled to explain scientific development purely in terms of social factors. Rather, a realist explanation of scientific development must make reference to complex interactions between social and natural factors, and these interactions will vary between different parts of science and at different stages of development of particular parts of science.

There are many reasons why a realist approach to the social construction of knowledge, and to the interpretative flexibility so evident in risk regulatory contexts, might provide an illuminating methodology (Radder 1992; Collier 1994; Sayer 1997). In particular, whereas scientistic analysts tend to be drawn toward accepting all of science as authoritative and socially neutral (or at least the science performed by socially dominant institutions), and relativistically inclined writers are drawn toward an impartiality (or at least a professional suspension of judgment) about the truth or falsity of different knowledge claims (prior to, during, and after their analysis of those knowledge claims), analysts presupposing a realist epistemology are able to appreciate that certain parts of science may be more or less reliable and robust than others. At least two investigative strategies can be used to empirically investigate that appreciation.

First, while empirical observation is often theory dependent, this does not entail that knowledge claims are entirely immune from empirical

verification. The various theories and beliefs implicated in particular observations and experiments are not necessarily part of the same theoretical and belief structure as that which is under scientific investigation. Consequently, choices between competing knowledge claims sometimes can be made by reference to empirical evidence in a nontautological and nonarbitrary fashion. In other words, we can sometimes discriminate between social practices that are likely to result in more as opposed to less reliable representations of the world, and between those different representations themselves.

Some published constructivist works on the science and politics of risk implicitly make judgments about more or less reliable scientific practices by reference to empirical evidence, although usually only as a critique of risk assessment in general. Wynne (1992) argued correctly, for example, that risk analysis is often far less reliable than its practitioners allege because regulatory scientists and government officials tend to depict risk assessments as if they were fully determinate and, thus, fail to acknowledge the complexity of, and their own level of ignorance about, real-world risks. For example, official and corporate assessments of the risks posed by chemicals are typically predicated on assumptions that exposure only occurs to one chemical at a time, that whatever indicator of damage chosen for assessment is a technical imperative, and that the behavior of human actors within a technological system is determinate and predictable, whereas in the real world risk problems rarely, if ever, comply with those models (Krohn and Weyer 1994; Wynne 1989, 1992). The models are intellectual abstractions adopted for pragmatic and opportunistic reasons. That critique (and the analysis in this article) rests on the realist claim that the world actually is sometimes more complex and contingent than prevailing (reductionist) intellectual models acknowledge. While this is a reasonable claim, it presupposes that we can, as policy analysts, privilege our interpretation of various items of empirical evidence that point toward complexity, indeterminacy, and variability in real-world risk problems as a sufficient basis upon which to problematize dominant institutional practices. As we argue below, this analytical tool can also be applied to specific knowledge claims and practices.

Our second investigative strategy involves making judgments about the relative reliability of knowledge claims and practices by reference to those standards and beliefs that we individually and collectively hold to be the most reliable and coherent. Although all scientific standards and beliefs are, at least in part, social products, some are nonetheless extremely robust whereas others are particularly fragile. Beliefs in certain types of logic or statistical inference, for instance, tend to remain relatively consistent both over time

and within and between scientific disciplines. Such standards provide a common set of benchmarks without which belief and communication would be impossible (Lukes 1982; Collier 1994). As policy analysts, we can reach judgments about the relative reliability of specific knowledge claims and practices by examining the extent to which they and their constructions are consistent with such standards and beliefs.

In the next section, those methodological strategies are applied in an analysis of U.S. and U.K. regulatory decision making about the toxicity and safety of a widely used group of pesticides. We argue that the U.K. assessment was not only less prudent, as most existing U.S./U.K. studies of regulation have indicated, but also less well constructed and, therefore, less reliable.

Ethylene Bisdithiocarbamates: Risk and Regulation

Ethylene bisdithiocarbamates (EBDCs) are a group of closely related chemicals that have been used extensively since the 1950s as fungicides on a wide range of food products (U.S. National Research Council 1987, 214). The most common EBDCs are mancozeb, maneb, metiram, nabam, and zineb (all various metal salts of ethylene bisdithiocarbamate). Several agrochemical firms including Rohm & Haas and Du Pont manufacture the compounds, which by the mid 1980s were generating approximately $500 million per annum in worldwide sales (U.S. National Research Council 1987, 209). Human exposure to EBDCs can and does occur, not only occupationally but also through the consumption of treated produce (Advisory Committee on Pesticides [ACP] 1990; U.S. Environmental Protection Agency [EPA] 1989).

In the late 1980s, the U.K. Ministry of Agriculture, Fisheries and Food (MAFF) and the EPA reviewed the use of EBDCs but reached different conclusions about the toxicity and safety of the fungicides. Most pertinent, the EPA (1989) concluded that a decomposition product, contaminant, and metabolite of EBDCs called ethylenethiourea (ETU) was a "probable human carcinogen." By contrast, MAFF concluded that there was no evidence that ETU posed any risk to human health (ACP 1990). Carcinogenicity risk assessment is a notoriously controversial procedure, and different scientists and regulatory authorities frequently disagree about the carcinogenic risk posed by particular chemicals (Nilsson, Tasheva, and Jaeger 1992). But how, in this case, did MAFF and the EPA reach such different conclusions about the carcinogenicity of ETU?

Toxicological Evidence

Five separate rodent-feeding studies had been conducted on ETU by the late 1980s. The first such study, on mice, was commissioned by the U.S. National Institutes for Health (NIH) in the late 1960s and found a statistically significant increase in liver tumors following dietary exposure to ETU (Innes et al. 1969). Two subsequent rat studies, commissioned by the U.S. National Cancer Institute (NCI) and the U.S. Food and Drug Administration in the early 1970s, reported significantly higher levels of thyroid tumors following dietary exposure to ETU (Ulland et al. 1972; Graham et al. 1975). In the late 1980s, two more feeding studies were performed by the U.S. National Toxicology Program (NTP), and statistically significant increases of liver tumors in mice and thyroid tumors in rats were discovered (Chhabra et al. 1992).

At the time of the EPA and MAFF reviews, at least 243 short-term genotoxicity tests had been completed on EBDCs/ETU. They were conducted both by public sector research institutions and by manufacturers of the fungicides, the latter taking the form of unpublished data submitted to regulatory authorities in support of product registration. About one-third of the tests reported a positive genotoxic response, whereas the remaining two-thirds did not. Genotoxicity evidence is important because it is widely accepted that any level of exposure to a genotoxic carcinogen may increase the probability of initiating carcinogenesis (Department of Health 1991; U.S. Office of Science and Technology Policy 1985).

Risk Assessments

In 1987, EPA scientists reviewed the toxicological evidence on ETU and concluded that rats had developed thyroid tumors experimentally as a result of a physiological mechanism that would not occur at the levels of ETU to which humans were being exposed (Sette 1990, 17). EPA scientists argued that the mechanism responsible for the mouse liver tumors was unknown and concluded that it would be prudent to extrapolate the findings in mice to humans and to assume that ETU was a liver carcinogen at any level of exposure. EPA scientists also argued that despite inconsistent results in the genotoxicity studies, EBDCs and ETU produced a spectrum of genotoxic effects, thus supporting a presumption of carcinogenicity (Dearfield 1988, 1989). In 1990, the EPA's advisory panel of external expert scientists endorsed the agency's judgment that ETU was a probable human carcinogen (Jaeger 1990).

In 1990, MAFF's Advisory Committee on Pesticides (ACP) (1990, 69) concluded that there was no evidence that either the EBDCs or ETU was

genotoxic. That interpretation differed from the EPA's judgment in part because the ACP omitted eighty-one of the completed genotoxicity studies from its review, fifty-one of which had been reported as showing positive evidence of genotoxicity. That pattern of selection resulted in greater neglect of the data reported as positive when compared with those reported as negative.[1] All 81 of the omitted studies had been published in the open literature. As one MAFF official explained:

> For [mutagenicity and related screening tests] to have relevance to the regulatory process they must follow protocols which have been fully validated, must have been carried out under rigorously controlled conditions and in accordance with the principles of good laboratory practice (GLP). All supporting data must also be available for inspection as part of the evaluation process. . . . Most studies reported in the open literature are not conducted for regulatory purposes, cannot be assumed to be GLP compliant and provide only a summary of the data generated. (MAFF Pesticide Safety Division, personal communication, 28 September 1993)

The implication was that less weight was assigned to studies that failed to meet those criteria than to those that did. By contrast, EPA scientists did not make the same distinction between published studies (as long as they had been peer reviewed) and unpublished studies (EPA Office of Pesticide Programs, personal communication, 18 March 1993).

Another reason for the difference between the two regulatory regimes' interpretation of the genotoxicity data was that the ACP defined a genotoxic risk to humans on the basis of studies conducted in in vivo test systems only, that is, tests on whole animals. Many, although not all, of the positive genotoxicity findings had only been reported in in vitro test systems, that is, tests on tissue samples. In vitro tests do not provide the normal supportive environment of the parent organism and, thus, do not allow functions such as repair mechanisms to operate. EPA scientists did not make the same distinction between in vivo and in vitro evidence (EPA Office of Pesticide Programs, personal communication, 18 March 1993).

The ACP did, however, agree with the EPA's conclusions about the likely irrelevance of the rat-feeding studies to humans, but the ACP's assessment of the mouse studies differed from that conducted in the United States. The ACP argued that the findings from the mouse studies were unlikely to be relevant to humans and offered several reasons why that might be the case. First, the ACP (1990, 72) suggested that mouse liver tumors may not always arise as a consequence of exposure to a chemical but, rather, may arise spontaneously. For example, the ACP's only comment on the NIH study was that "all these tumours occur frequently in mice and the increased incidence is unlikely to

be compound related" (p. 19). The ACP also suggested that "most" chemicals that are nongenotoxic (as ETU was assumed to be) and cause increases in liver tumors also cause "hepatocellular necrosis [cell death in the liver] or a sub cellular change such as peroxisome proliferation" (p. 73). Both hepatocellular necrosis and peroxisome proliferation can themselves cause cancer; that is, they are secondary or indirect mechanisms. The implication was that exposure to ETU would not pose a risk to humans because peroxisome proliferation does not occur in humans, and hepato- cellular necrosis occurs only after continuous and high levels of exposure to a chemical. Finally, a small proportion of the liver tumors observed in the NTP study were of an unusual type that is rare in humans and may be linked to estrogen stimulation in mice (p. 73).

The ACP (1990, 73) strongly implied that the mouse liver tumors occurred due to one or more of those factors and insisted that the significance of the mouse data to humans was therefore uncertain. On that basis, together with the apparent lack of genotoxicity, the ACP assumed that any adverse effects of ETU would occur only above a "threshold" dose and that levels of exposure below that threshold posed no carcinogenic risk to humans (p. 76).

How Do Experts Contrive to Disagree?

The existing literature on cross-national risk regulation suggests that U.K. regulators tend to require far more substantial and direct evidence of harm before classifying chemicals as hazardous than regulators in the United States and elsewhere. That essentially informal commitment, it is suggested, is manifest not only in the precise location of burdens of proof but also in a number of more "technical" differences, for example, in the standards used to certify evidence and the models used to analyze data (Gillespie, Eva, and Johnston 1979; Brickman, Jasanoff, and Ilgen 1985; Jasanoff 1987b; Abraham and Millstone 1989; Wynne 1992; Millstone 1997).

One gloss on this case study might be that the ACP's standards for certifying the relevant evidence, and for deciding how great a burden of proof the evidence could and should sustain, were simply more exacting than those deployed by the EPA. Specifically, the ACP appeared to be more sensitive than the EPA to the possibility that in vitro genotoxicity test responses would not provide information relevant to whole organisms such as humans and the possibility that ordinary peer review mechanisms would not be as relevant for, and perhaps not as rigorous as, regulatory review. Furthermore, the ACP appeared to have been more cautious than the EPA in classifying ETU as a direct-acting human carcinogen given an assumed lack of genotoxicity, the

absence of causal evidence of a direct mechanism for mouse liver carcinogenicity, and several postulated mechanisms that either operate indirectly or are unrelated to ETU exposure. While this is a plausible explanation of regulatory divergence, we want to argue that different informal levels of precaution do not fully explain the different risk assessments.

Selection of Genotoxicity Data

If one were to assume that the ACP and the EPA's different criteria for certifying the relevance of the genotoxicity evidence were equally credible, albeit more or less exacting, one might nevertheless expect those standards to be applied consistently. For example, although eighty-one published genotoxicity studies were omitted from the ACP's review, an additional eighty-seven published studies were nonetheless included, and one would expect that those selected published studies would have met the ACP's nominal selection criteria. The evidence shows, however, that this was not the case.

For example, fifty of the published studies on ETU listed as being fully analyzed during and informing the ACP review were conducted by laboratories under the auspices of an International Collaborative Programme (ICP). The studies were subsequently published in a report that provided details of the findings from each participating laboratory (deSerres and Ashby 1981). The ACP's review document contains at least two specific errors because the text refers to one in vitro chromosomal aberration test and one sperm abnormality test when in fact two of each of those tests were conducted under the ICP and reported in the published ICP documentation (deSerres and Ashby 1981, 551-59, 712-20). What is interesting about those errors is that although they were not made by the study authors, each of whom wrote a chapter in the ICP document detailing their findings, exactly the same errors were introduced in summary tables contained within chapters that themselves summarize and collate the results from the individual chapters (deSerres and Ashby 1981, 78-79, 88-89). That strongly suggests, therefore, that the ACP relied on the summary tables for its review rather than the individual chapters (which clearly document all the tests), let alone full supporting data that might conceivably have been obtained from each laboratory. The ACP was, therefore, willing to violate its own alleged criteria of selection.

Furthermore, several of the published genotoxicity studies that were omitted from the ACP's review (ostensibly because they failed to meet suitable protocol and reporting requirements) were subsequently deemed to be relevant and acceptable by the ACP when, on different occasions, it reviewed the safety of other types of pesticides. This is understandable, since many of the

published studies tested more than one pesticide at a time using the same protocol. For example, two of the tests omitted by the ACP were part of a study conducted by Georgian et al. (1983) that investigated the genotoxicity of the herbicide alachlor as well as one of the EBDCs in two different test systems (an in vitro test on human lymphocytes and an in vivo test on rat bone marrow cells). The ACP (1991, 67-68) conducted a review of alachlor during the late 1980s that included the Georgian et al. study and certified it as reliable and relevant. It is interesting that Georgian et al. reported a positive response in the in vivo genotoxicity test with EBDCs, a result that directly contradicts the ACP's (1990) specific claim that "there was no evidence that ETU or any of the EBDCs were genotoxic in vivo" (p. 76). Such inconsistencies again suggest that the criteria MAFF claims to adopt when selecting genotoxicity evidence were, at least in part, merely a post hoc explanation for a different set of selection practices.

Interpretation of the Mouse Studies

One of the ACP's claims was that the mouse liver tumors were likely to have arisen spontaneously. Yet, the ACP failed to examine the historical control data for the relevant mouse strains, as recommended by most regulatory authorities when suspicions about spontaneous results arise (International Programme on Chemical Safety 1990; EPA 1986; Department of Health 1991). Historical control data can be used to help assess whether concurrent controls provide a typical species/strain pattern of spontaneous tumors and, thus, whether a reported statistical increase in commonly occurring tumors could be a false positive result. As one expert group (convened by the U.S.-based Nutrition Foundation) noted:

> The difficulty in assessing whether the incidence of mouse hepatoma is due to treatment relates to both statistical and biological questions concerning the results of any given test. The problem is somewhat diminished if the incidence in treated animals is clearly higher than concurrent controls. The evidence is even more persuasive if the incidence in treated animals is also higher than historical controls and/or is dose related. (Doull et al. 1983, 30)

One of the mouse strains used in the NIH study (and the strain used in the NTP study) was the hybrid $B_6C_3F_1$ mouse.[2] That strain has been used routinely in studies performed by the NCI. In nearly 200 NCI studies, the incidence of liver tumors in control $B_6C_3F_1$ mice ranged from 18 percent to 47 percent in males and from 2.5 percent to 8 percent in females (Doull et al. 1983, 18). The actual incidence rates in the NIH and NTP studies approached or reached 100 percent in both sexes.[3] Those rates massively exceeded the

tumor incidence in the NCI historical control data, particularly for the female mouse, as well as in concurrent controls. On the basis of the two studies, and the historical control data for that strain of mouse, in combination with robust principles of statistical inference, there is a clear prima facie case for arguing that the ACP's assertion about the likelihood of spontaneous tumors is unreliable and unconvincing.

The ACP also suggested that necrosis or peroxisome proliferation might have indirectly caused the mouse liver tumors. In the mid 1980s, Rohm and Haas conducted several research projects designed to provide an understanding of the mechanism of ETU-induced mouse liver carcinogenesis. One of those projects investigated ETU's effects on peroxisomal proliferation but found no evidence of such an effect (Fenner-Crisp 1989, 3). That study was not mentioned by the ACP. Furthermore, as the EPA (1989, 2:13) noted in its review of the NTP study, "cellular necrosis was not observed in any group"—a point that yet again the ACP neglected to mention. It is not clear why the ACP was invoking either of these mechanistic hypotheses unless it either had not looked at the available evidence or because it wished to create the impression that there was more uncertainty about ETU-induced liver carcinogenesis than was actually the case.

By assuming that a dose threshold existed for the carcinogenic effects of ETU, the ACP used a less cautious approach than that stipulated by the Department of Health's (1991, 64) Committee on Carcinogenicity, which recommends that where no mechanism has been established, nongenotoxic carcinogens should be assumed to have no threshold. Although MAFF officials claim to follow the committee guidelines (MAFF Pesticide Safety Division, personal communication, 19 February 1993), the departure from those guidelines in the case of ETU is evidently an example of a more general phenomenon. The minutes of a meeting between EPA staff and officials from the European Community, in connection with both the United Kingdom's EBDC review and risk assessment practices more generally, noted that

> if carcinogenic, but not genotoxic [the European Community uses] the NOAEL/safety factor/ADI approach to regulation that the U.S. uses for non-carcinogens, because [it is an advocate] of thresholds in such cases. As noted above, [it] simply must demonstrate that a threshold mechanism is "*plausible*." (Ives 1991, 4, emphasis added)

As far as the ACP's assessment of ETU was concerned, plausibility apparently applied to conjectures that were deemed acceptable not only in the complete absence of supporting evidence but also when contradicted by available evidence.

During the U.S. review, EBDC manufacturers confronted EPA scientists with similar arguments about the likelihood that mouse liver tumors might be an indirect effect of ETU exposure. However, EPA scientists did not believe that industry had any rational scientific argument with which to explain the induction of the ETU-induced liver tumors (EPA Office of Pesticide Programs, personal communication, 18 March 1993), and the EPA (1989) noted that "no supportable or adequate scientific argument has been put forth to persuade the Agency to alter its current policy [of assuming that ETU was a direct carcinogen]" (p. 2:19). Indeed, industry scientists themselves conceded finally that there was no evidence in support of a mechanistic argument that could explain the formation of those mouse liver tumors (Flamm 1989).

The Blackwell-Smith Study

It is useful to contrast the ACP's claim that the mouse liver tumors observed in studies on ETU were likely to have arisen spontaneously with its assessment of one of several long-term feeding studies that had been performed on zineb, one of the parent EBDCs. That study, called the Blackwell-Smith et al. (1953) study, involved feeding groups of ten rats six different doses of zineb for two years. The study reported tumors in the dose groups and the control group, but there was no significant difference in the incidence between treated and control groups.

In the early 1970s, the EPA (1989, 2:2) reviewed the Blackwell-Smith study and decided that because of the small number of animals in each dose group, it did not constitute sufficient evidence of a negative carcinogenic response—a decision that continued to be upheld in the late 1980s. As the EPA (1982, 2:11) pointed out, with one of ten animals in each of the Blackwell-Smith study's control groups developing tumors, there would have to be a minimum of six of ten animals in the treated groups developing tumors before a statistically significance difference could be observed. Moreover, even if the true cancer rate in the treated groups were 60 percent compared with 10 percent in the control group, the number of animals in the study was so low that only a 66 percent chance of detecting the effect existed (EPA 1982, 2:11). In other words, the likelihood of a false negative result with this study was high, even if it was being tested with an extremely powerful carcinogen. The ACP made no comment on the reliability of Blackwell-Smith's findings and effectively treated them as negative evidence of carcinogenicity.[4] Thus, while the evidence from the ETU mouse studies indicated that for statistical reasons a false positive result was unlikely, the evidence from the Blackwell-Smith study indicated that the probability of a

false negative result was really quite high—precisely the opposite interpretation the ACP arrived at.

Summary

The above account suggests that the divergent U.S./U.K. assessments of carcinogenic risk occurred only in part as a consequence of different, informal attitudes to uncertainty, as reflected in the divergent standards used to certify and interpret evidence. The U.K. assessment also involved modes of reasoning and knowledge claims that were contradicted by available empirical evidence and/or defied basic, widely agreed upon understandings of logic and statistical inference. In particular, it included certain genotoxicity evidence (that was generally supportive of its reassuring narrative on safety) even though that evidence did not meet its own selection criteria. At the same time, other genotoxicity evidence that elsewhere fulfilled the ACP's supposed criteria of selection (but undermined the ACP's chosen narrative) was excluded. The ACP assessment also discounted the relevance of evidence from feeding studies for reasons that not only were speculative but also contradicted by available evidence. In other words, the U.S. assessment was a relatively robust construction whereas the U.K. assessment was altogether more fragile.

Explaining Regulatory Divergence

U.S. Risk Assessment: The Creation of More Robust Constructions

It is commonplace to characterize U.S. regulatory cultures as formal, adversarial, legalistic, and open (Jasanoff 1990). While that characterization may underestimate the scope that exists for more informal and private forms of regulatory negotiation, there are, nevertheless, comprehensive powers of judicial and legislative oversight; legally enshrined rights for third parties to obtain extensive information on, and challenge, risk regulation; and powers for the judiciary to arbitrate over, and stipulate procedures for, scientific and administrative decision making. In the latter case, for example, the courts have insisted that regulatory agencies make many assumptions and inconsistencies in regulatory decisions explicit, disclose their methodologies, respond to contradictory evidence, and support conclusions in a manner capable of judicial understanding (Jasanoff 1992, 203).

As sociological accounts of U.S. risk regulation have shown, the powerful political demands generated within the U.S. polity to diminish, or at least

continually supervise and challenge, administrative discretion has meant that scientific analysis—one of the few warrants of official authority—is forced into playing a far more prominent role in policy making than would be the case in many other countries. At the same time, however, scientific knowledge is constantly prone to processes of deconstruction as competing actors seek to challenge particular policy predispositions (Rushefsky 1985; Jasanoff 1987b). As Brickman (1984) put it, U.S. institutions "both place science on a pedestal and work to knock it off" (p. 110).

Although, partly for these reasons, U.S. science policy cultures consequently generate severe problems of political legitimacy (Jasanoff 1990) and regulatory inefficiency (Mendeloff 1988), they have nevertheless managed to constrain certain types of interpretative flexibility to the extent that knowledge claims and practices that are overtly noncredible are unlikely to survive the regulatory process. During the EBDC review, for example, many hundreds of publicly available documents were produced detailing the interpretation of each piece of evidence, as well as many of the procedures involved in reaching an estimate of risk. On both procedural and substantive grounds, third parties could have challenged, and sometimes did challenge, the EPA's risk assessment. None of this can ensure that EPA scientists will not depart from stated inference principles or deploy noncredible modes of reasoning, but it does make it far more unlikely.

Although the institutionalized distrust between actors and institutions in U.S. regulatory cultures is widely recognized, what is less well appreciated is that some aspects of that mistrust have been instilled through a continuous process of social learning about how and why regulatory practices were sometimes hindering the protection of public health. For example, before 1970 the U.S. pesticide-licensing regime, then operating under the aegis of the U.S. Department of Agriculture (USDA), enjoyed wide discretion over when and how its powers to refuse or revoke pesticide licenses should be exercised. In practice, the USDA did not ask manufacturers for toxicological data and only in extreme circumstances did it attempt to obstruct the sale of pesticides (Bosso 1987). Carson's *Silent Spring* first began to make public these regulatory weaknesses in 1962, and this culminated in 1970 with responsibility for pesticide regulation moving to the newly created EPA and the establishment of a more adversarial, legalistic, and open form of control (Hoberg 1990). Several of those shifts in administrative culture were provoked by the recognition on the part of the judiciary that restricting EPA discretion and subjecting expert analyses to a full and public airing could minimize the "capture" of the new agency by industrial interests (MacIntyre 1985).

Then, in the early 1980s, a congressional subcommittee discovered that several of the safety evaluations submitted to the EPA by manufacturers did not identify or discuss toxic effects that were indicated by the raw data or contained highly questionable arguments that sought to minimize the significance of adverse findings (U.S. Congress 1983, 13, 121).[5] EPA scientists informed the committee that they only had sufficient resources to detect and pursue a small proportion of findings that were evident in experimental data but had been ignored or dismissed in submitted reports. Even more disturbing, the subcommittee discovered that some EPA evaluations were nothing more than verbatim transcriptions of summaries submitted by the manufacturers (U.S. Congress 1983, 124).

Sociologists of science have long recognized that scientists have to take on trust much of what they believe to be true. As the U.S. Congress discovered, however, high levels of trust can entail that overtly noncredible knowledge claims become certified via the regulatory process. Congress thus set in motion policy changes that institutionalized distrust—for example, by providing more resources for the EPA to deter and act as a deterrent against what one of the subcommittees called "shoddy science" and by introducing checks and balances on the data evaluation process. Thus, during the EBDC review, both submitted evidence and the EPA's assessment of that evidence was subject to internal and external peer review. These procedures by no means diminish all conflict over how evidence should be interpreted, but they do help to avoid the certification of overtly noncredible knowledge claims.

U.K. Risk Assessment: Was It Well or Poorly Constructed?

There are considerable differences between U.S. and U.K. regulatory policy cultures. In the United Kingdom, regulatory institutions operate in a highly discretionary and discreet fashion, and this has given rise, in the pesticides arena and elsewhere, to an informal, negotiated decision-making process subject to few explicit or legally binding rules or procedures (Brickman, Jasanoff, and Ilgen 1985). High levels of legally sanctioned official secrecy, together with weak or ineffective powers of oversight by third parties, the judiciary, and even Parliament, hinder wider public accountability and allow pesticide decision making to take place in private within collusive social networks. The scope for exercising administrative and scientific discretion in U.K. policy processes is consequently enormous. These institutional factors, among others, are typically drawn on in the science studies literature to explain why U.K. risk assessment is sometimes more conservative than it is in other countries (Gillespie, Eva, and Johnston 1979; Jasanoff 1987b). Most analysts assume, however, that U.K. risk assessment is generally a robust and

credible practice, albeit a conservative one, or are at best ambivalent about such questions.

A refusal on the part of MAFF officials to allow interviews with those civil servants directly responsible for reviewing the toxicological evidence on EBDCs means that any explanation of why aspects of the ACP's assessment were less than credible must remain provisional. Three hypotheses suggest themselves in this context. In descending order of attractiveness, they are (1) that the ACP selected and evaluated evidence in a shoddy and unsystematic fashion, and that it is merely a coincidence that it showed a marked tendency to assess evidence in ways that favored an overall evaluation of minimal risk; (2) that the ACP accepted a third party's shoddy and/or biased evaluation of the relevant evidence without checking that account for its reliability; and (3) that the ACP selected and evaluated evidence in a systematically biased fashion. It is difficult to avoid the conclusion that at least one of these hypotheses must be correct. Yet, because both submitted evidence and internal deliberations are, as a matter of policy, not disclosed by MAFF, both the toxicological and regulatory arguments advanced by the fungicides' manufacturers in support of the U.K. regulatory review and the deliberations that occurred within the regulatory process remain concealed. It is, however, clear that there are few incentives to ensure that U.K. regulators do not conduct sloppy, uncritical, or opportunistic assessments of toxicological evidence.

Not only is discretion enormous and accountability and transparency severely limited, but potential conflicts of interest between regulators and industry are procedurally unacknowledged. Unlike the United States, the United Kingdom has not, in the case of pesticide regulation, separated institutional sponsorship of the agricultural and food industries from responsibility for public and environmental health, nor does it place safeguards on the movement of personnel from regulatory authorities directly to the regulated industry, or the holding of industrial consultancies by expert advisors. In fact, a close alignment of interests between MAFF and the industry it regulates have historically been integral to the operation of the regulatory regime. Before 1985, pesticide regulation in the United Kingdom took the form of a poorly resourced voluntary agreement between government and pesticide manufacturers and distributors. Partly because MAFF relied on the industry to police the scheme, good relations were vital for the success of the regulatory procedure. As one senior official in MAFF's pesticide safety division noted in 1978: "Unreasonable demands [for product and toxicological data] could lead to a break in the *essential mutual trust* between government and industry" (Bates 1978, 174, emphasis added). This type of mutual trust may, for example, diminish the extent to which MAFF considered it necessary to provide sufficient resources and expertise to scrutinize submitted data or

conduct rigorous regulatory assessments. In 1985, the last year in which U.K. pesticide regulation operated on a nonstatutory basis, and just prior to the EBDC review, only twenty professional staff were employed in MAFF's Data Evaluation Unit (Environmental News Data Services 1986). Although MAFF recruited additional staff, in 1989, just before the EBDC review was completed, the British Agrochemical Association (BAA), trade unions, and several environmental groups jointly called for additional resources for MAFF's Data Evaluation Unit. The BAA's director claimed that the Harpenden Laboratory "just hasn't got enough people and enough skill to cope with these reviews without bringing everything else to a full stop at the same time" (Environmental News Data Services 1989, 3). In the 1990s, more staff members were recruited, although MAFF had to begin contracting out parts of its data evaluation due to insufficient in-house expertise (MAFF Pesticide Safety Division, personal communication, 19 February 1993).

The relevant independent advisory committee, the ACP, appears to be far less pivotal than most commentators suggest or assume. The bulk of each technical assessment, including draft regulatory recommendations, is conducted by civil servants within MAFF. A scientific subcommittee, also predominantly comprising civil servants from MAFF and other government departments, then reviews the draft and, as MAFF (1992) put it, "after resolution of any scientific issues raised by data holders the [scientific subcommittee's] recommendations, amended if necessary, go forward to the [ACP]" (p. 3). The task of the ACP, however, "is not to carry out a detailed scientific assessment of the data but to set the analysis carried out by the [scientific subcommittee] in a wider context" (House of Commons 1987, 61). Thus, many of the crucial decisions concerning the selection and interpretation of evidence are made by civil servants. While the ACP may in principle be able to review a scientific assessment in detail and gain access to the original data, that is not its normal or routine role.[6]

To summarize, most of the crucial decisions about the selection and interpretation of toxicological evidence are conducted by departmental civil servants at least in part through a process of negotiation with pesticide manufacturers. This process occurs in a context where high levels of trust between MAFF and the agrochemical industry are institutionalized; discretionary potential is high; accountability is low; and oversight by the legislature, the courts, and the wider public is virtually zero. Furthermore, detailed scientific oversight by the relevant independent expert committee appears to be relatively low, at least for routine decision making. These features do not entail that risk assessments will not be robust, but little within the culture of U.K. regulation appears able to facilitate the detection or prevention of less than credible scientific practices or judgments. Indeed, the evidence presented in

this article suggests that U.K. regulatory cultures and institutions can and do allow the production of knowledge, as used to support the protection of public and environmental health, to be compromised in ways that are far more difficult to sustain within U.S. regulatory cultures.

Pesticide regulation will probably always involve a tension between agricultural and industrial production and the protection of public and environmental health. In the United Kingdom, there are fewer institutional procedures than in the United States for managing those conflicts and for learning about, and responding to, the different ways in which those conflicts manifest themselves in practice. Public health in the United Kingdom would benefit from an institutional separation of pesticide regulation from sponsorship of the agricultural industry, greater regulatory oversight, a greater degree of transparency, and more opportunities for wider participation in regulatory decision making. In the wake of the bovine spongiform encephalopathy/Creutzfeldt-Jakob disease saga in the United Kingdom, the Food Standards Agency was created specifically in response to MAFF's conflict of interest in food policy. Yet, the government proposes to leave pesticide regulation to MAFF, apparently on the grounds that the Pesticide Safety Division is highly efficient and effective. The evidence presented here indicates that such a degree of confidence is unwarranted.

Conclusions

Few constructivist analysts interested in risk would defend an epistemological perspective in which scientific knowledge is viewed solely as an epiphenomenon of social practice; heuristically, however, most constructivist analysts tend to approach their studies and deconstructions of science as if that were precisely the case. Questions about the relative validity or truth of competing knowledge claims, and forms of knowledge production, are therefore avoided, although some constructivists might argue that that lacunae is unimportant because those questions are sociologically uninteresting. Our view, on the other hand, is that sociological questions are not the only interesting ones, and that questions of validity and truth are, at least partly, sociological, and interestingly so.

We have argued that while a sociological deconstruction of scientific discourse should start from a point of view that suspends judgment as to the truth of the claims at issue, once that analysis has been completed, it may well be possible and desirable to abandon that suspense of judgment and, thereby, make an active, sociologically illuminated contribution to the debate in which the scientists were themselves contenders.

One of the failings for which scientistic analyses of official expert discourse were rightly criticized in the 1960s and 1970s was that they were deliberately deferential to the most authoritative institutions producing knowledge for policy, thereby marginalizing and denigrating nonofficial, lay, and/or critical accounts of risk. To the extent that constructivist analyses give equal credence to the "underdog," they have provided an improvement on the previous approach, but strict post hoc agnosticism (that might be either deliberate or a symptom of timidity) prevents commentators from making judgments that discriminate between better and worst accounts of some aspect of reality. Once we have learnt how some sets of scientific claims have been constructed, we can sometimes determine which ones have merely been shoddily or opportunistically constructed and contrast them with others that are demonstrably more robust. Just as scientistic analyses are prone to leaving the credibility of influential institutions unchallenged, so extreme constructivists may uncritically provide credibility to knowledge claims that deserve to be discredited.

Furthermore, whenever one steps beyond the limits of academic sociology to engage with science policy, one inevitably makes judgments about which knowledge claims, experts, or institutions one deems to be more or less credible. Because that is unavoidable, the intellectually and politically honest approach is one that explicitly acknowledges that such judgments are being made and endeavors to support those judgments by reference to the scientific and sociological evidence.

Sociological students of science are, therefore, potentially well placed to make a positive contribution to policy analysis and policy making. Analyses based on realist sociological deconstructions of competing knowledge claims can make a positive and critical contribution to science, sociology, and policy. Efforts to understand how well or poorly scientific claims have been constructed are valuable because they can tell us something about the risks that are being assessed and about the beliefs of scientists that the scientists and policy makers themselves may be unable or unwilling to acknowledge.

Perhaps one of the single most exciting benefits of such an approach, for those concerned with public policy making as well as with an academic agenda, is precisely that the more we appreciate how well and how poorly constructed particular scientific claims are, the better placed we become to articulate a research agenda and strategy that, given appropriate time and resources, might enable the research community to contribute to a diminishing of some of the most policy-relevant scientific uncertainties. That benefit would not solve all of the problems faced by policy makers, but it might simplify them, and would be beneficial to the scientific community and to society

in general; however, it can only accrue from the adoption of a realist framework.

Notes

1. Of the published genotoxicity data on ethylene bisdithiocarbamates, 85 percent of the studies that reported a positive genotoxic response were omitted compared with 42 percent of the studies that reported a negative response. Similar proportions of positive and negative data on ethylenethiourea that had been published were omitted. All unpublished data were included in the Ministry of Agriculture, Fisheries and Food's review (van Zwanenberg 1996, ch. 7).

2. One of the strains in the National Institutes for Health study was a cross between C57BL/6 and C3H/Anf mouse strains (Innes et al. 1969, 1102). This produces the $B_6C_3F_1$ hybrid (A. Kocialski, Environmental Protection Agency, personal communication, 6 September 1994).

3. In the National Institutes for Health study, 14 of 16 (88 percent) of the male $B_6C_3F_1$ hybrid mice that had been fed a diet containing 646 parts per million of ethylenethiourea (ETU) developed liver tumors compared with 3 of 14 (21 percent) of the male controls, whereas 18 of 18 (100 percent) of the treated female $B_6C_3F_1$ mice developed liver tumors compared with 0 of 18 (0 percent) of the female controls. Both results in treated mice were significantly different from the controls (Environmental Protection Agency 1979, 14). In the National Toxicology Program study, 45 of 46 (98 percent) male $B_6C_3F_1$ hybrid mice that had been fed a diet containing 1,000 parts per million of ETU developed liver carcinomas compared with 19 of 47 (40 percent) male mice that had been fed a diet containing 333 parts per million of ETU and 13 of 43 (30 percent) male controls. In that study, 47 of 49 (96 percent) female mice that had been fed a diet containing 1,000 parts per million of ETU developed liver carcinomas compared with 29 of 50 (58 percent) female mice that had been fed a diet containing 333 parts per million of ETU and 2 of 49 (4 percent) female controls. The results for male mice in the top dose group and for female mice in both dosed groups were significantly different from those for the controls (Sette 1990).

4. The Advisory Committee on Pesticides's [ACP] (1990) review document did not state explicitly that the Blackwell-Smith study provided negative evidence of carcinogenicity, but one can reasonably assume that it was treated as such because (1) the ACP later concluded that all of the adverse effects observed in tests on the fungicides could be attributed to ethylenethiourea (ETU); (2) the Blackwell-Smith study was the only long-term feeding study on zineb that was reviewed by the ACP; and (3) unlike the actions of the Environmental Protection Agency (EPA), no call for additional long-term feeding studies was made to resolve uncertainties about the question of ethylene bisdithiocarbamate (EBDC) carcinogenicity. By contrast, the EPA (1989, 2:2) concluded that there was as yet insufficient data to reach a conclusion about the risks posed by the parent EBDCs.

5. The congressional committee noted, for example, that "one current member of the Toxicological Branch says that, while he thought he had encountered 'every trick in the book' during his career evaluating chronic toxicity experiments and data, he has recently been amazed by new levels of 'ingenuity and cleverness' employed by some pesticide registrants. He expressed admiration for the capacity of registrants to advance new arguments minimizing the significance of negative experimental findings" (U.S. Congress 1983, 121).

6. Indeed, G. Hollis, head of the Ministry of Agriculture, Fisheries and Food's (MAFF) Pesticide Safety Division from 1989 to 1991, confirmed that the Advisory Committee on Pesticides's role is largely one of legitimating the work of MAFF civil servants. In 1998, Hollis told a U.K. public inquiry into the bovine spongiform encephalopathy saga that "I speak with knowing what happens with the Advisory Committee on Pesticide, when they consider all the evidence

that had been put up to them by individual civil servants going through a great deal of work. Those civil servants individually are experts in their field, they are top people on toxicology and so forth, but they have to have their findings gone over by an independent body of people and they give essentially a stamp of approval. If those civil servants wrote papers, they would not have that credibility."

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